Australian & New Zealand Society Of Pediatric Dentistry

IN THIS ISSUE

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PRESIDENT'S REPORT

Dear Colleagues,

During the last three months the Society has been involved in a number of projects.

On the local scene John Brownbill and his Policy Committee have produced a policy document detailing a number of issues that affect the dental health care of our children. This is initially for your information and comment and discussion and acceptance will not occur until the next Bi-annual meeting.

In September the Society is sponsoring a postgraduate programme in Sydney. Dr. Richard Widmer is co-ordinating its development and as it is being held in conjunction with I.A.D.R. the meeting should provide an intensive programme in paediatric dentistry. More information later in the Newsletter.

The 12th Congress of the International Association of Dentistry for Children was held in Athens from the 1st to 5th June 1989. The Congress was well attended with 350 registrants. There were 25 representatives from Australia and an enjoyable five days were had by all.

During the meeting I represented the Society at the I.A.D.C. Council meeting where many issues were discussed. Firstly the Association will change its name to the International Association of Paediatric Dentistry and this will take effect from 1991 at the Kyoto meeting.

The second important topic was the delivery of the Newsletter and Journal. There has been a new editor appointed so that it is hoped that a Newsletter will arrive in the very near future.

Thomas Moleer presented the report of the Task Force established during Roger Hall's reign into the future of the Association and Paediatric dentistry. The Report discussed many issues and particularly the future involvement of young paediatric dentists worldwide. This will be discussed in the next letter.

Faithfully yours

JAMES LUCAS

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SECRETARY'S REPORT

At the I.A.D.C. Conference in Athens, the Council elected a new editor, an Englishman, Dr. Barry Scheer of the London Hospital Medical School.

Thus, in future we will receive quarterly international newsletters, published in Britain and paid for from our Federal subscriptions. The first issue will be in September this year.

However, a new international paediatric dentistry journal is mooted, probably at first in conjunction with the British Dental Journal for which we will have to each individually apply and pay an extra ten pounds sterling.

I'll keep you informed about the progress of the journal, but I feel it will be some time before it comes to fruition.

In the meantime, moves have been made to ensure that our own excellent Newsletter is published on time so as to keep us up to date with the latest developments at home.

JOHN KEYS

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EDITOR'S NOTES

The title of the Newsletter has caused concern with paediatric being printed as "Pediatric" instead of "Paediatric". This will be corrected in future issues.

R E S O U R C E L I B R A R Y

Each State has developed a variety of resources over the years. These can be videos, i.e. Nursing Bottle videos from South Australia, or pamphlets or books. Could a list of each State's Resources please be send to -

Dr. Alain Middleton
Suite 2/6 Old Castle Hill Road
Castle Hill N.S.W. 2154

"FACILITATING NATURAL HOST PROTECTION OF TEETH: THE WHOLE STORY ABOUT SALIVA"

This is an edited version of a Presentation by Dr. Mark Jensen from the School of Dentistry, University of Iowa U.S.A. (This article is reproduced with the permission of the Wrigley Company)

The positive effects of saliva, essentially, are a host protective mechanism that are responsible for oral clearance, lubrication and swallowing. Also, increasing the optimum function of taste in our oral cavity. But fundamentally saliva is a component of the host that is really responsible for protection against dental caries, not just deglutition or swallowing.

We can think of this process in an interesting way because Backer-Dirks in the 1960's examined white spot lesions in a group of eight year old children, documented them and followed these children over a period of seven years. So in looking at them at the age of fifteen he examined the white spot lesions and surprisingly out of 72 white spot lesions after 6 years, 37 or over half of these had actually reversed themselves entirely, becoming sound surfaces without evidence of visual white spot lesion. Unlike what some of us had been taught earlier, only 9 of these, a much smaller percentage over 7 years, had actually progressed to a cavitated lesion that required restoration.

Now this is a pretty remarkable finding in that it begins to point us in the direction of thinking of dental caries as an interaction between these three factors requiring plaque and carbohydrate to produce acid for demineralisation and loss of calcium phosphate from the tooth structure. Instead of just thinking of it as a progression of the disease that once we see it start, it's a matter of stepping in and doing a cavity preparation and restoring that surface, we now need to think this process is really an equilibrium process. It's a balance between demineralisation which occurs maybe many periods during the day and remineralisation. Calcium and phosphate from saliva being placed back into the tooth's surface via reprecipitation, recrystallisation. In other words, repair of the process, so that it can actually at the earlier stages of the white post situation, be reversed entirely.

Now when we think about this process there are lots of ways to continue that and demonstrate it intraorally with both root and enamel sections. Basically, we think of enamel caries as an acidogenic process ... the acidogenic theory of dental caries. Under this situation, we really are talking about the duration of acid exposure being related to the amount of mineral loss.

If we're to think of a dietary pattern or an idealised one in which there are three balanced meals, we could think of three periods of demineralisation and the periods between meals, we could think of these periods as remineralisation and we could have a balance situation.

Unfortunately, most individuals do not eat in that manner. When we think about children in the U.S., some groups snack fermentable carbohydrates up to 18, 22 and in some cases maybe 30 times a day during the waking hours. Under these circumstances, we may eliminate the period of remineralisation ... we may have essentially a continuous acid exposure situation of demineralisation and it's not suprising that we see a diet related disease ... when they abuse, we see dental caries.

Although the Viperholm study and Hopewood House study definitely shows this is a clinical way that caries are related to diet, we can measure something short of producing the disease process in human volunteers and that is what Stephan did in the late 30's and early 40's, looking at plaque acids. This can be done in a variety of ways either with sampling of the plaque, touching electrodes to the surface, or a little more sophisticated way first done in Zurick, Switzerland by Hans Graffin employing pH telemetry ... either by wire or by radio telemetry. Under these conditions of measurement of acid in the oral cavity we see similar patterns, but when the plaque is measured by pH telemetry at the plaque/enamel interface, we see on the undersurface that instead of a twenty minute acid attack or a short exposure there is a much lower pH. There is more acid produced at the internal surface of plaque and it is a more prolonged response than we see by other methods.

We have essentially measured this process, as have people in many other laboratories in the world, and found that a number of the snack foods that are used frequently by individuals throughout the world are extremely acidogenic, such as muesli bars and some of the fruits like raisins and fruit bars. Mothers may select these for their children because they really feel they are healthy. In fact, when these foods are taken between meals, we know from pH telemetry that these may be some of the worst exposures of acid ... the most prolonged in the oral cavity we could possibly have.

We can look at a whole spectrum of these foods and it turns out that in looking at hundreds and now thousands of foods there are always some surprises. For example, even the consumption of some fresh fruits and vegetables such as apples, cause acid exposure that may contribute to dental caries.

When we look at the literature, it's not very surprising because there is fermentable carbohydrate there and the micro-organisms and plaque are capable of fermenting it, and producing acid in the oral cavity which would cause demineralisation. Even some other systems such as the rate animal model do sow us historically that foods like apples even, are capable of producing some dental caries.

If we're to think of these foods that do promote caries as being taken or eaten at meal times, we'd not be as concerned. What we'd really like to do is counsel our patients to eliminate or change that pattern of eating fermentable carbohydrates between meals - have them eat at meal times and make those changes.

Unfortunately, behavioural changes are very difficult to make. We could then say that an additional way that helps balance this relationship anyway is to take a direction that would be removal of plaque. Let's balance that relationship by essentially eliminating some of the acid formation by removing plaque. So we say "brushing should be done frequently after meals" (that's what we all recommend). I'm not sure what we mean by "frequently" but essentially, if we could have our patients brush after meals and thoroughly remove that plaque from the tooth surface it would have an impact on dental caries. This is probably regardless of the therapeutic effect of fluoride dentifrice which is a much more powerful chemotherapeutic tool.

When we look at plaque pH readings from interproximal electrodes, we see that after an acid exposure and plaque has accumulated there, brushing with any of the leading dentrifices for a three minute period has very little, if any, effect on that interproixmal acid exposure. Then we can say "O.K. We understand the proximal site is very difficult to reach - we'll just have our patient floss".

It turns out that if we can get our patients, for periodontal reasons, to floss once a day, we'd be very lucky. So to actually ask that patient to floss after snacks and meals would be very unrealistic and I think we would have few patients who actually comply and carry out a regime such as this.

We can see that plaque does accumulate in a proximal site. If we have a fermentable carbohydrate, say a jelly donut, we experience a prolonged exposure of acid at the proximal site for several hours below a pH for minimal phosphate dissolution.

It turns out that looking at both chewing paraffin and chewing a surgarless gum can very rapidly stimulate saliva and reverse this challenge in a very short period of time. We looked at a five minute chewing period and a ten minute chewing period and this plaque pH that would be a prolonged extended attack, can be returned very rapidly in a ten minute period to the original resting values and in a five minute period to above a safe level o by chewing paraffin in our saliva.

This tells us something very important about the protective effects of saliva and the delivery of it in a mechanical way to the very inaccessible proximal site. The buffer capacity of the saliva when it is stimulated is much higher and the flow rates are dramatically higher. Unfortunately, if we just had a rinse, we would not reach this inner proximal site and that's one of the reasons, even after a topical application of fluoride in the dental office, we have to go back into the oral cavity and floss to those sites to deliver even a thixotropic fluoride gel to the inaccessible proximal site. But chewing paraffin or sorbitol containing gum (a sugarless gum) mechanically pumps the saliva to those areas, neutralising the acids which are produced as well as perhaps clearing any remaining fermentable carbohydrate.

It has been reported in several publications now that by chewing a sugarless gum for a ten minute period after a carbohydrate snack will neutralise plaque acids as well as assist in the clearance of these acids and of the substrate. It could be done with wax but we don't have many patients that would choose to do that.

Saliva is stimulated and pumped to those hard to reach proximal surfaces and actually helps balance this process of demineralisation. So, if we were to think about reducing the risk of caries, we would still recommend that the patient brush frequently with a fluoridated dentifrice. Also, that they floss properly each day the way that we teach them, to remove plaque, and to reduce the frequency of snacks. Lastly, the natural host protective mechanism of saliva could be facilitated by chewing gum after these snacks to neutralise acids that have been produced.

Actually, in thinking about this, we have really begun to learn more of the whole story of saliva which is responsible for balancing this situation, clearing both substrate and acids from the cavity and protecting us against the progression of the caries' process.

When we think a little bit harder about this balance between remineralisation, it's not surprising that when we look at the acidogenic potential of meals, we have an extended challenge time just to our normal meal pattern alone. So, if we're to look at some of the original data produced by Thomas Imfeld in Zurich, most meals have fermentable carbohydrate and result in the same acid exposure of several hours to this interproximal site.

We decided to continue this investigation and begin to look at what we could do after acid production from these meals. So we looked at five different normal meals in the U.S. - these meals were actually provided in our hospital staff cafeteria in conjunction with one of the registered dieticians. We came up with five different meals; the first meal being a roast beef and potato meal; the second, a sandwich (a sloppy joe with a bun) the third a tuna and pasta noodles meal; the fourth a chicken and rice meal; and the last one is a hot dog with a bun. It turns out that all five of these meals created the prolonged acid exposure that we talked about earlier, with something like a danish sweet roll or the jelly filled donut. So we had several hours of that acid exposure from each of these meals and we discovered if we eliminated the dessert at the end of the meal it made no difference.

Now, from what we know about chewing gum and the snack foods, we said "Why not recommend chewing sugarless gum after meals".

So we designed an experiment to use these five meals and to look at a sugarless gum to be chewed after each meal. I guess it's not very surprising that a chew of the sugarless gum after the meal helps clear that food rapidly and acts in the same way as with the snack foods that were investigated earlier.

So really we've facilitated that natural host protective mechanism and we'd want to recommend that we reverse that acid attack to balance the situation: prevent demineralisation and facilitate the process of remineralisation. So we believe we can recommend chewing gum after the meal.

Now that's not the whole story in that lots of individuals would rather chew a sugar-containing gum than a sugarless gum for various reasons. Some patients just don't like a sugarless gum. For many individuals that is not a problem, and we'd certainly recommend that that type of gum could be used.

What we decided to do was to look at two sugar containing gums after the meal. The amount of sugar contained in the gum relative to the amount of sugar in the meal and especially compared to the fermentable carbohydrate in the meal is very small ... in fact miniscule compared to the rest of the fermentable carbohydrate.

When the test group chew a sugar gum after the meal we saw a pretty surprising outcome. Retrospectively, it's not surprising. But essentially, the two sugar containing gums, one of stick form , and one in a pellet form were really no different when chewed for 20 minutes than sugarless gum.

Both resulted with all the individuals in all five meal patterns in the complete reversal of that interproximal plaque acid attack. So during that 20 minute period, it's indistinguishable from sugarless gum eaten after a meal and the explanation turns out to be relatively simple. What we are looking at is a period of maybe only 3 minutes or 3 - 5 minutes in which the sugared gum essentially becomes flavoured gum base. In a short period of time, the carbohydrate is chewed out, we have sugarless gum if you will, or a gum cud that mechanically stimulates saliva together with chemical stimulation with flavour etc. that is responsible for stimulating flow rates. Saliva, with a higher buffer capacity is in a very important way delivered to the inaccessable site reversing the acid challenge.

These studies which are soon to be published in the British Dental Journals tell us that if we're going to recommend that we balance this situation of demineralisation, what we can recommend is that this can be rapidly reversed by chewing gum for a 20 minute period.

We essentially cannot distinguish between the sugarless gum or the sugar containing gum in stick or pellet form. So although we would like to recommend that we have no acid exposure whatsoever, that's impossible in a normal dietary situation.

So what we'd like to do is say for those patients who like the sugared gum or for a patient who has a risk at all or any evidence of white spots or a low salivary flow, is that in addition to all of the other means of balancing this process, in other words, normal flossing and brushing, optimal use of fluorides, meticulous oral hygiene etc., we could say that an easy way of taking advantage of this natural host protective mechanism of saliva would be to recommend that they chew gum after meals.

Chewing gum is one additional step that can be taken to balance this caries equilibrium process, to help prevent more demineralisation or get less demineralisation and increase the process of remineralisation to overall reduce the risk of caries formation or progression.

So we've really come a long way in terms of some basic science knowledge, some understanding of the physical chemistry of this process to say that we are looking at a pretty complex situation. But then we can make some practical clinical recommendations, especially for patients who like to chew gum. Chewing gum would seem a very sound way of balancing this process. So we'd like to give this advice in a reasonable way especially to those patients with an increased risk such as the ones we talked about earlier that may be taking medications that reduce salivery flow. We now have a means to stimulate what flow there is and delivering it to the caries prone sites.

In this way, I think that we can put together our training and apply it in a very practical way. We can deliver that information to our patients and actually help place an impact on preventing the disease by actually reversing the stages before we do a surgical approach in those patients who have a continual experience of a new cavitated lesion at one or six month recalls.

We can now look at that and say we have an additional understanding of the disease and we may be able to balance that relationship back to the symbiotic situation via the normal protective mechanisms of saliva. One way to do that is to add chewing gum to the normal dental recommendation and put it in the proper prospective so that it can be used between meals to reverse the carbohydrate challenge to the dentition from various snacks. Chewing sugared or sugarless gum after meals, could become part of normal dietary practice.

If we can't get them to floss in those areas right after eating, chewing gum for a 20 minute period after eating meals could be recommended.

I hope that I have been able to contribute one small piece of information that may have been helpful to you and your patients. I would like to encourage you to continue to stay updated in understanding this disease process. As you will be able to assist your patients to manage their caries problem in a sound and preventative way.

Fig I

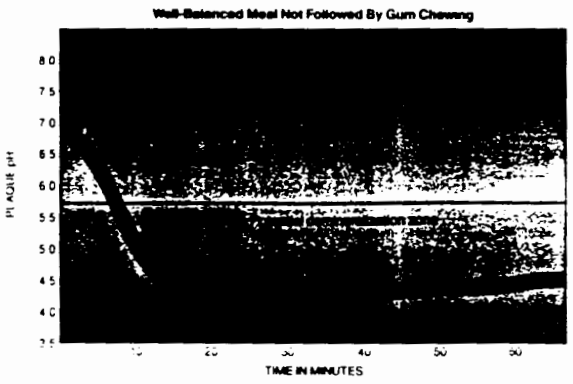
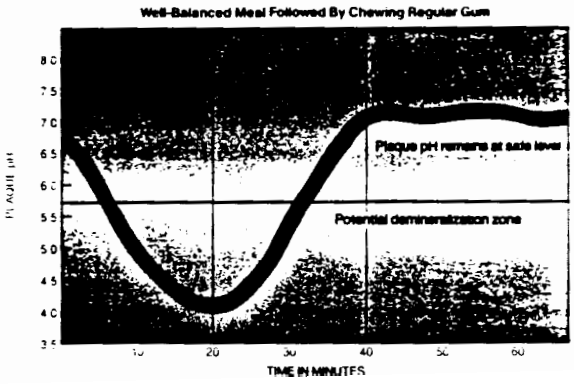


Fig II



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Kashket et al: Reduction in Demineralisation Resulting from time at which the chewing of sorbitol gum commences after sucrose challenge. Journal Dental Research 68 (3) 1989

EDITOR'S NOTE: In most discussions of the effects of saliva emphasis is placed on acidogenicity of food and the effect of saliva. Whether this acidogenicity is the same as the cariogenicity is one aspect of this topic that must be developed.

POSTGRADUATE PROGRAMMES

28 - 29 SEPTEMBER 1989

The Federal Branch of the Society is sponsoring a programme for postgraduate students at Westmead Hospital.

The two-day programme will consist of firstly, clinical seminars which involve the examination, assessment and treatment planning of a variety of patients exhibiting a variety of medical and physical disabilities. The postgraduates will be associated with the paediatric dentists attending and hence allow intensive and extensive discussion of the clinical problems.

The second day will consist of seminars on a range of topics including cleft palate care, paediatric restorative dentistry and treatment of medically compromised children.

The programme is being organised by Dr. Richard Widmer with the co-operation of the Westmead Dental Hospital. I recommend this programme to all postgraduates as it will allow you to meet other postgraduate students, share experiences and also meet and discuss paediatric dentistry with those who specialise.

The Federal Branch of the A.N.Z.S.P.D. is giving $100 towards the airfare of each attending postgraduate student and if accommodation is a problem do not hesitate to contact Dr. Richard Widmer at Westmead who may be able to arrange some accommodation at the Hospital.

It is hoped that this programme can become a calendar event for our Society.

James Lucas
President
A.N.Z.P.D.

.... P O L I C Y D R A F T ....

These Policies are for your information and comment. Please address all communications to the Editor.

POLICY OF THE AUSTRALIA AND NEW ZEALAND SOCIETY OF PAEDIATRIC DENTISTRY

1. ORAL HEALTH PROGRAMMES

Oral care is an essential part of total health care and we emphasize the importance of prevention.

2. EMERGENCY CARE

Emergency dental care for children should be available at all times.

3. PREVENTIVE DENTISTRY

The Australia and New Zealand Society of Paediatric Dentistry endorses and supports proven procedures and techniques of preventive dentistry and urges that they be given high priority in practice.

(i) FLUORIDATION

The Australian and New Zealand Society of Paediatric Dentistry affirms that fluoride in community water supplies and in other proven preparations provides a safe and effective means of reducing dental decay. The adjustment of the fluoride levels in community water supplies to optimal concentrations is the most beneficial and inexpensive method of reducing the occurrence of dental decay. Alternative fluoride programmes should be initiated in areas not served by fluoridated water supplies and the dosages should not exceed the recommendations of the National Health and Medical Research Council.

(ii) PRENATAL FLUORIDE SUPPLEMENT

Lacking sound scientific evidence, the Australia and New Zealand Society of Paediatric Dentistry does not endorse the use of prenatal fluoride supplements.

(iii) MOUTHGUARDS

The Australia and New Zealand Society of Paediatric Dentistry endorses the use of professionally-fitted mouthguards in body contact sports and hazardous recreational pursuits. User-fitted mouthguards generally have inadequate retention to be effective. The Australia and New Zealand Society of Paediatric Dentistry urges all junior sports organizations and schools to make the wearing of professionally-fitted mouthguards compulsory in body contact sports and hazardous recreational pursuits.

(iv) ORAL HYGIENE

The Australia and New Zealand Society of Paediatric Dentistry encourages the development of tooth brushing with a soft brush from the time of eruption of the first tooth. From as early age as possible the child should learn effective plaque removal with brush and floss, but the parent or care-giver should assume responsibility for oral hygiene procedures until it can be demonstrated that the child can effectively and safely perform the procedures.

4. DENTAL CARIES

(i) DIET

Caries is related directly to the frequency of refined sugar intake and to the length of time cariogenic substances remain in contact with the teeth. The Australia and New Zealand Society of Paediatric Dentistry strongly recommends voluntary restriction of the consumption of beverages and foods of a confectionery nature. The Australia and New Zealand Society of Paediatric Dentistry urges that school administrators eliminate the sale or provision of cariogenic foods in school canteens.

(ii) SEALANTS

Providers of dental care for children should recommend the placement of sealants on all non-carious susceptible pits and fissures of recently fully erupted permanent teeth.

(iii) NURSING BOTTLE CARIES

Prolonged bottle feeding of infants can lead to "nursing bottle cavities". Infants should be taught to drink from a cup before their first birthday. In particular the use of juices in a bottle is particularly damaging.

5. RADIOGRAPHS

Radiographs should be taken according to individual patient need to maximize the yield of diagnostic information and minimize ionizing radiation exposure.

6. INFANT DENTAL CARE

Infant dental care begins with prenatal counselling. For optimal caries preventive effect, dietary fluoride must be present within a few months of birth; and the diet must be restricted in refined carbohydrates.

Policy Committee
John Brownbill, Chairman 5 April 1989

BRANCH NEWS

QUEENSLAND

The Queensland Branch held two meetings on 6th February and 2nd May last.

At the first meeting the Guest speaker was Dr. Dorothy Radford, Deputy Director of Cardiology - Prince Charles Hospital. Her topic was "Congenital Cardiac Problems in Children".

Dr. Radford delivered a most interesting presentation on this subject. Major congenital cardiac lesions were enumerated with further discussion on Subacute bacterial Endocarditis. Left to Right Shunts included Ventricular Septal Defects, detected by a murmur which in about half the cases fail to close spontaneously, and therefore require surgery. Atrial Septal Defects, more difficult to detect because of absence of a murmur, can be picked up by E.C.G. and should be closed by age five. Patent Ductus Arteriosus is an indication for surgical legation in all cases.

Stenotic lesions include Pulmonary Stenosis, Aortic Stenosis and Coarctation of the Aorta. The most common type of Right to Left Shunts is Tetralogy of Fallot.
Diagnosis of Endocarditis include such systemic signs as malaise, fever, new or changing murmur, splenomegaly, anaemia, cultures, haematuria, and embolic phenomena. This is a lethal condition.

Prophylactic antibiotic cover is an essential consideration.

At the Second Meeting the Guest speaker was Dr. Bill Young, Senior Lecturer, University of Queensland Dental School. Dr. Young's topic was Clinical Oral Pathology in Children.

Dr. Young presented a lecture focusing mainly on Giant Cell lesions which may be either central or peripheral. Hormonal and inflammatory stimuli play a role in the changes of bony architecture seen in these lesions.

Clinically the Peripheral Giant Cell Granuloma occurs as an exophytic, often bluish brown coloured lesion on the gingiva or alveolar process.

Histologically it consists of giant cells in a fibrous connective tissue stroma associated with the mucoperiosteum and active osteoblasts with new spicules of bone being formed in the tumour.

Central Giant Cell Granulomas occur centrally in bone and appear as a multilocular radiolucency.

Other giant cell containing lesions of bone have been associated with hyperparathyriodism, cherubism. and the aneurysmal bone cyst. Hyperparathyroidism exhibits familial hyperparathyroid activity and is not common in children. Cherubism exhibits hereditary (Autosomal dominant) proliferation in jaw bones of giant cells in a connective tissue stroma which can disturb the occclusion. Radiographically there are large polycystic spaces in the maxilla and mandible.

The aneurysmal bone cyst appears as a bluish mass often with swelling over the area of bone involvement. Histologically multinucleated giant cells are present in a vascular fibrous connective tissue stroma. The bone pattern appears like Chinese characters. Radiographically the borders of the radiolucency is scalloped. Aspiration yield a straw coloured fluid.

A report on the meeting held on 5th June 1989 and the Branch's Clinical Weekend held on 12-13 August will appear in the next Newsletter.

The October meeting of the Branch will be held on 2nd October 1989 - Dr. A. Moule - Endodontic Management of the Traumatized Immature Tooth.

Christopher Ho - Secretary/Treasurer

VICTORIA

The Branch held its first dinner meeting for 1989 on February 23rd. Our guest speaker, Professor Mark Walqvist, delivered the inaugural Professor Elsdon Storey Memorial Lecture. Professor Walqvist has just been appointed to the Chair of Medicine at Monash University.

In his lecture entitled "Nutrition, Medicine and Dentistry" he made many references to Professor Storey as both a friend and a colleague.

He discussed the important relationship between social activities and health; and the increasing interest in non-nutrients in food which have biological activity, such as caffeine, salicylates, phyto oestrogens and opioids. He highlighted the problems in the methodology of biomedical research: the need to shift from single factor testing to a more integrated approach. He suggested that the factors which will have the most impact on nutrition, medicine and dentistry in the future are molecular biology, technology and the environment.

Professor Walqvist concluded his lecture with a tribute to Professor Storey, describing him as a man of vision, goals, perspective and integrity.

Mrs. Pat Storey presented Professor Walqvist with a gift to commemmorate the inaugural Professor Elsdon Storey Lecture.

The Second meeting of the Branch was held in April and the Guest speaker was Dr. Roger Warne, the Regional Geriatrician, University of Melbourne, who gave an informative and entertaining lecture entitled "Geriatric Medicine - Prevention and Practice". He drew attention to the shift in average age of the population and the consequent challenge for health care workers. The dentist's role in maintaining an adequate dentition is essential, because of the importance of masticatory function to nutrition, and thus to general health.

Arrangements are underway for the Annual Convention to be held in association with the Australian Society of Orthodontists (Vic. Branch) at Leonda, Hawthorn on 27th and 28th October. The speaker will be Dr. Robert Little, D.D.S., M.D.S., Ph.D., Professor of Orthodontics, University of Washington. The title of his presentation is "Contemporary Orthodontic Strategies for the General Practitioner, Paediatric Dentist and the Orthodontist".

Mary Ellen Wilkinson - Secretary

NEW SOUTH WALES

This year we have held two very well attended meetings. The first was on Tuesday 21st March and 26 members and guests enjoyed Rosemary Stanton, our Guest speaker, deliver an interesting lecture - "Eating for Peak Performance". This was followed by our meeting of May 23rd, where 37 members gathered for dinner and to hear Dr. Uta Schroder deliver a presentation on "Calcium Hydroxide: When? Why? and How?"

A report on the seminar "Afternoon of Updates in Paediatrics" held in conjunction with the R.A.H.C. on 21st July will be included with the next Newsletter.

Jill Arnena - Secretary

WEST AUSTRALIA

The programme for the year began in grand style, when a group in excess of thirty gathered for the first meeting at A.D.A. House on 12th April. Branch President, John Hands, welcomed members and their guests and then introduced the Guest Speaker, Dr. Jack Goldblatt. Dr. Goldblatt is the Director of Genetics at the Princess Margaret Hospital for Children, a position he has held for just over a year. Dr. Goldblatt then proceeded to deliver a most entertaining and informative lecture, a lecture well supported by suitable slides and by references to actual cases, some which have presented in Perth and others which have been featured in movies. All present were soon aware that the speaker was quite a movie buff!

Dr. Goldblatt emphasised the difference between genetic and congenital conditions. Some genetic conditions, in fact, may not be apparent at birth, but manifest later in life e.g. Huntingtons Chorea. Congenital conditions are present at birth but are not necessarily genetically determined.

Dr. Bill Brogan proposed the vote of thanks, and in addition to providing an appropriate conclusion to proceedings, he certainly summed up the feelings of all in attendance in congratulating Dr. Goldblatt on his fine presentation.

After the highly successful two day course in 1988, "Margaret River Revisited", a similar country course was held on 21st and 22nd July with the topic - "Orthodontics for the General Practitioner". The presenter was Dr. Mithran Goonewardene, an orthodontist both in private practice in Perth and with a half time position at the University of W.A.

Alistair - Secretary

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"FROM THE JOURNALS" - with John Burrows

MARGINAL RIDGE STRENGTH OF RESTORED TEETH WITH MODIFIED CLASS II CAVITY PREPARATIONS

The restoration of the interproximal carious lesion has always been a challenge for the practitioner. Although most of the destructive effects of dental caries can be diagnosed and restored directly, the location of an interproximal lesion can present problems. In the early stage of development, the proximal lesion is surrounded by unaffected tooth structure that blocks access to the lesion. The intact marginal ridge and tooth structure occlusal to the lesion are routinely removed.

Recently, a modified Class II cavity preparation has been introduced for the treatment of interproximal carious lesions in which the entire cavity preparation is placed through an occlusal access opening. This preparation preserves the tooth's marginal ridge and its proximal contact area. While minimizing the amount of tooth structure lost, the effect of this preparation on the integrity of the preserved marginal ridge is unknown. This study specifically measured the resistance to fracture of the marginal ridge in teeth prepared with the modified Class I cavity preparation. The effects of adhesive (composite resin) and non-adhesive (amalgam) restorative materials when used in this preparation were also evaluated.

The results showed the fracture resistance of the marginal ridges of teeth prepared and restored using this modified cavity was not significantly lower than that of intact teeth. Without restorative materials placement in prepared teeth, marginal ridges were significantly weaker. Amalgam and composite restorative materials, were similar in their ability to reinforce tooth structure.

(COVEY D., SCHULEIN T.M. & KOHOUT F.J. JADA 118 Feb 1989:199-202)

CUSPAL REINFORCEMENT IN PRIMARY TEETH: AN IN-VITRO COMPARISON OF THREE RESTORATIVE MATERIALS

Twelve primary second molars were evaluated using a repeated-measures protocol for cuspal deflection stress following restoration of MOD preparations with either a composite resin, amalgam, or glass-ionomer restorative material. Ranking the materials for support of cuspal strength, then composite resin was best with glass-ionomer next and then amalgam. These reports are consistent with those reported for permanent teeth.

(DONLY K.J. WILD T, & JENSEN M.E. Paediatric Dent. 10(2) 102-104)

A CLINICAL RADIOGRAPHIC, AND SEM EVALUATION OF CLASS II COMPOSITE RESTORATIONS IN PRIMARY TEETH

Children aged between 8 and 12 years having a primary molar with interproximal caries, and occlusal and approximal contacting teeth were used in this study. All restorations were placed with local anaesthetic and rubber dam. A conventional Class II cavity preparation was cut with exposed dentine walls protected with Dycal. The enamel margins were etched using a fine brush for 69 seconds. A stainless steel matrix band was placed and the cavities were filled with condensable composite. Two increments were used

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with a final light cure following removal of the matrix band, and a second group had the cavity filled with a bulk placement of the resin cured for 60 seconds following contouring and a final light cure following removal of the matrix band.

The restorations were evaluated at baseline, six months and one year for surface appearance, colour match, marginal adaptation, marginal discolouration, anatomic form, and secondary caries. The children were encouraged to return exfoliated teeth, or preferably, to return when the tooth was mobile. 58 of the original 60 restorations were available for assessment.

The clinical evaluation showed high success rates of the Class II composite restorations after one year. Radiographic examination after one year, however, demonstrated that in 40% of the restorations radiolucent defects could be seen at the gingival margin; as exfoliation of these teeth was anticipated in the near future, no replacement was done. the SEM evaluation of buccal lingual and cervical margins showed the highest percentage of defects to be at the cervical margin, with no difference found between the two filling techniques.

The approximal wall is not amenable to direct clinical examination; when exfoliated teeth were examined, it was found that the surface and margin of the approximal areas fell far short of the excellent results of the occlusal area.

(EIDELMAN E, FUKS A, & CHOSAK A. Operative Dent. 1989, 14:58-63)

INFLUENCE OF MILK AND FOOD ON FLUORIDE BIOAVAILABILTY

Aqueous solutions of NaF and sodium monofluorophosphate (MFP) were given to fasting young adults, and plasma F levels were measured by use of a F-ion sensitive electrode. The subjects were given tablets containing 2mg F, either as NaF or as MFP, under different experimental regimens:

(a) on a fasting stomach
(b) together with milk, or
(c) together with breakfast and milk.

Plasma peak levels were reduced when the tablets were taken together with food. Intake of milk reduced F availability by 30% compared with the fasting stomach; this effect was abolished when milk was taken as part of the breakfast.

It is suggested that formation of Ca salts and entrapment of F in coagulation products of milk are important factors causing reduction of F availability, and that prolonged stay of the chyme after concomitant ingestion of food allows F to become liberated from bound forms and coagulation products by digestion processes.

(TRAUTNAR K & EINWAG J. J Dent Res 68(1):73-77,Jan 1989)

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THOUGHTS

Men are more careful of the breed of their horses and dogs than of their children. William Penn

There is no finer investment for any community than putting milk into babies. Winston Churchill

No one ever keeps a secret so well as a child.
Victor Hugo